Neonatal ECMO: Neuroimaging and Neurodevelopmental Outcome

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Intracranial injury continues to be a major complication associated with extracorporeal membrane oxygenation (ECMO)-treated neonates. The reported frequency of abnormal neuroimaging has ranged from 28% to 52%, depending on neuroimaging techniques and methods of classification. The purpose of this chapter is to describe types of imaging techniques commonly used to evaluate the ECMO neonate, to specify different types of injuries that have been reported, and to identify factors which increase the risk of injury. We will then describe the functional impact at age 5 years following neonatal brain injury among ECMO infants.

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Risk Factors

Multiple factors increase the risk of intracranial injury in infants undergoing extracorporeal membrane oxygenation (ECMO).1-6 In addition to the necessity for anticoagulant use during bypass, all candidates for ECMO have suffered from hypoxemia and acidosis, many with evidence of vasomotor shock. Hypercarbia, volume expansion, pneumothoraces, and blood pressure fluctuations alter cerebral blood flow.1,7 Hypotension before or during ECMO may result in cerebral ischemia. Using near infrared spectrophotometry, Liem and coworkers demonstrated changes on ECMO including increased cerebral blood volume, loss of autoregulation, reactive hyperperfusion, and hemodilution.6 Arterioles that are already maximally dilated may, in the presence of impaired autoregulation, rupture because of increased blood flow pressure.9,10 Diminished pulsatility with venoarterial ECMO may also affect cerebral blood flow. Abnormal venous drainage resulting from jugular vein ligation has also been implicated as a cause of cerebrovascular injury subsequent to stasis within periventricular medullary veins.11

After unilateral carotid ligation, infants on ventilatory support have been noted to have decreased cerebral blood flow.9 In a study by Hunter and coworkers, cerebral blood flow of lambs was measured by laser Doppler flowmetry during venoarterial (VA) and venovenous (VV) ECMO. Carotid ligation resulted in a decrease in the cerebral blood flow (CBF) of the right cerebral cortex. However, this decrease was only transient (60 seconds), with elevation of cerebral resistance. Using VV ECMO, no change in CBF was observed.12

There has been concern that ligation of the carotid artery may cause lateralized cerebrovascular injury. Several early small series noted an increase in injuries to the right hemisphere in infants who underwent ligation of the right carotid artery.13-15 In larger series by Adolph and coworkers and Bulas and coworkers, however, no lateralization of lesions was noted by neuroimaging.6,16 In a series of 355 infants using ultrasound (US), computer tomography (CT), magnetic resonance imaging (MRI), or clinical evaluation, Graziani and coworkers also demonstrated no selective or greater injury to the right hemisphere as compared with the left.17 In a cohort of 31 infants treated with ECMO evaluated by MRI, there was no lateralization of major brain lesions.18 No lateralization was noted in a group of infants studied by cerebral proton magnetic resonance spectroscopy following ECMO.19 However, focal brain lesions were significantly associated with an asymmetric cerebrovascular response to carotid ligation of the right versus left middle cerebral artery as detected

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by Magnetic Resonance Angiography (MRA) \((P < 0.5)\). Schumacher and his colleagues\(^5\) have argued that no lateralization of lesions among ECMO-treated neonates is indicative of increased vulnerability of the right hemisphere, since reports of intraventricular hemorrhage (IVH) and stroke in non-ECMO patients indicate relative vulnerability of the left hemisphere.

It is believed that premature infants on ECMO are at high risk of intracranial hemorrhage due to the presence of a friable germinal matrix with poor supporting stroma.\(^1,2\) Despite the exclusion of small premature infants from ECMO, studies have shown that younger infants continue to have a statistically significant increased risk of hemorrhage, even though the mean age was 38 weeks and only 27% of the hemorrhages originated in the germinal matrix.\(^6\) Although regions of ischemia are at risk for hemorrhage when heparin is used, infarcts in term infants weighing over 3 kg typically do not progress to hemorrhages.

Infants with sepsis are also at high risk for intracranial hemorrhage likely due to additional problems with coagulopathy.\(^20\) In a series by Hardart and coworkers, gestational age, sepsis, coagulopathy, and acidosis were all associated with a higher incidence of intracranial hemorrhage.\(^21,22\) Dela Cruz and coworkers demonstrated that elevated ACT and low platelet count were also associated with an increase in intracranial hemorrhages.\(^23\)

Infants with long circuit runs, particularly those with congenital diaphragmatic hernia, have the highest rate of major nonhemorrhagic lesions. With more conservative use of heparin, the risk of partial venous occlusion and microemboli has been shown to increase.\(^6,24\)

Widened interhemispheric fissures have been well described in infants on ECMO, with rates of occurrence as high as 59%.\(^1,23\) (Fig. 1). Rubin and coworkers (1990) believed this dilation was an intracranial manifestation of generalized edema.\(^26\) Other authors have suggested that increased sagittal sinus pressure associated with internal jugular vein ligation and cannulation of the superior vena cava is the cause of this dilation due to decreased cerebrospinal fluid resorption of the arachnoid vili.\(^11,27\) Widened extra axial space can develop as well, with severe cases noted following superior vena cava thrombosis.\(^28\) V V ECMO is particularly prone to decreased venous drainage resulting in an increased incidence of dilated interhemispheric fissure and prominent subarachnoid space.\(^29\) Due to the potential risk of venous stasis, cephalic drainage has been developed in an attempt to prevent neurologic complications by maintaining normal cerebral blood flow and increasing ECMO oxygen delivery.\(^30\)

**Cerebrovascular Imaging**

**Ultrasound**

Ultrasound is particularly useful in the evaluation of infants on ECMO due to its portability and lack of ionizing radiation. The presence of a large intracranial hemorrhage is a contraindication for ECMO initiation, thus a screening examination before cannulation is critical in the assessment of potential therapeutic options. Ultrasound has been sensitive in the evaluation of large cranial hemorrhages. In a series by Bulas and coworkers, sonography successfully identified 46 (94%) of 49 major intracranial hemorrhages, lesions that most affect the way an infant is managed acutely.\(^31\) Identification of isolated subependymal hemorrhages has been shown not to be at risk for progressive and should not prevent the initiation of ECMO therapy.\(^32\)

As the risk of hemorrhage is greatest in the first few days of ECMO, daily cranial sonograms have been recommended for the potential identification of a developing bleed. The question as to how often daily sonograms should be performed...
has been reviewed by several centers. In 1996, Biehl found daily sonograms cost effective only during the first 3 days on ECMO. In their series, 50% of intracranial hemorrhages occurred in the first 24 hours, 75% by 48 hours, and 85% within 72 hours of initiation of bypass. Further sonograms were deemed unnecessary unless there was a change in neurological status or multi-organ failure. Khan and coworkers suggested performing cranial sonograms for the first 5 days on ECMO unless a clinical suspicion was raised.

Large parenchymal hemorrhages are usually identified sonographically as focal regions of increased echogenicity (Fig. 2). With less than 30% of hemorrhages developing in the germinal matrix, it is crucial to look carefully within the peripheral parenchyma and posterior fossa for unusual regions of increased or decreased echogenicity. If a question-able lesion is identified, close follow up is useful as these bleeds often increase rapidly in size. Hemorrhages may appear hypoechogenic due to decreased coagulation.

In a series of 117 infants with sonographic or CT evidence of hemorrhage, 64% were parenchymal with 8.5% extraxial. The most common site for a parenchymal bleed was the cerebellum (27%). Cerebellar hemorrhages can be difficult to identify by ultrasound via the anterior fontanelle. A transmastoid view or imaging via the posterior fontanelle may improve the sensitivity of identifying these hemorrhages sonographically.

Ultrasound is less successful in identifying nonhemorrhagic lesions. Generalized edema can be difficult to differentiate from normal, and Doppler tracings are not useful in the assessment of autoregulation as pulsatility is diminished on VA bypass. Despite these limitations, early screening has been used to identify infants with severe edema. Von Allmen and coworkers noted that infants with evidence of severe edema on pre-ECMO sonograms had a 63% rate of occurrence of subsequent major intracranial complications (Fig. 3). Infarcts, small parenchymal hemorrhages, and extraaxial collections may not be visible sonographically. Sonographic distinction between hemorrhagic and ischemic lesions is difficult, as both can be echogenic. Numerous studies have demonstrated the superiority of CT and/or MRI in the identification of nonhemorrhagic and small hemorrhagic lesions. CT contributed additional information in 73% of neonates with intracranial abnormalities in a series of 286 infants screened by US and CT. Seventeen of these were major lesions not identified by ultrasound. Garber and coworkers noted that of 6.8% ultrasounds reported to be normal, abnormalities were identified by CT.

CT/MRI

Follow-up CT/MRI scans provide additional information in 72% to 93% of ECMO patients initially scanned with ultrasound. In a series of 130 infants with nonhemorrhagic abnormalities, the majority of abnormalities missed by sonography were classified as minor. However, 6 infarcts, 3 diffuse periventricular hypodensities, and 5 moderate atrophies were demonstrated only by CT. Other series have also demonstrated that CT is particularly useful in identifying infarcts, diffuse edema, and atrophy in infants with normal-appearing sonograms.

MRI can provide unique information at follow-up evaluation. MRA has demonstrated asymmetric cerebrovascular response to carotid ligation of the right versus left MCA. Cerebral proton magnetic resonance spectroscopy has been used to assess potential changes in brain metabolism following ligation. It was hoped that single photon emission computed tomography (SPECT) could show deficits not seen by neuroimaging. However, in a series by Kumar and coworkers, a normal SPECT scan was likely to predict normal outcome but an abnormal SPECT did not predict abnormal outcome. In a recent study using proton MRI and spectroscopy, nine neonates were evaluated following ECMO and no difference in right or left basal ganglia were noted, suggesting that ligation of the carotid artery did not produce persistent changes in brain metabolism in the basal ganglia in this small group. Larger studies have not been reported to date. As diffusion weighted imaging and higher Tesla scanners become more available, further information may become available for the assessment of ECMO-related cerebrovascular injuries.

Clearly, the type of injuries incurred varies to a great extent. In an attempt to elucidate the functional significance of the neuroimaging abnormalities, we developed and later modified a scoring system of injury severity and used this as a grouping variable (none, mild, moderate, severe) (Table 1). Using logistic regression, we demonstrated that the presence of neuroimaging abnormality by CT scan is the strongest predictor of long-term outcome.
Table 1 Representative Lesions by Severity on Routine Neonatal Neuroimaging

<table>
<thead>
<tr>
<th>Severity</th>
<th>Lesions Description</th>
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<tbody>
<tr>
<td>Mild (NIS = 0.5-3.5)</td>
<td>Wide interhemispheric fissure, Mild ventricular dilatation, Large subarachnoid space, Subependymal hemorrhage, Scattered petechial hemorrhages, Small extra-axial hemorrhages</td>
</tr>
<tr>
<td>Moderate (NIS = 4.0-6.0)</td>
<td>Single large parenchymal hemorrhage (&gt;1 cm), Patchy periventricular leukomalacia or hypodensity, Mild generalized atrophy, Combination of mild hemorrhagic and non-hemorrhagic abnormalities</td>
</tr>
<tr>
<td>Severe (NIS &gt; 6.0)</td>
<td>Moderate to severe generalized atrophy, Diffuse periventricular leukomalacia, Multiple large parenchymal hemorrhages (&gt;1 cm), Large parenchymal infarct</td>
</tr>
</tbody>
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Neurodevelopmental Impact of Perinatal Brain Injury

Given the relatively high rate of neuroimaging abnormality among ECMO-treated neonates, neurodevelopmental outcome studies have been important in defining the functional impact. Substantial outcomes research in other populations following perinatal brain injury underscores both neurospecificity and neuroplasticity in the young developing brain. Earlier studies of neuroimaging and outcome in the preterm infant serve as an important model for the ECMO-treated neonate. The introduction of routine cranial ultrasounds for preterm infants, rather than relying on CT scan for clinical indications, identified an unexpectedly high rate of “silent” hemorrhages; that is, injury not associated with handicap. However, when neurodevelopmental outcome measures were more specific than the designation of handicap versus no-handicap and extended beyond the infant/toddler period, more subtle neuropsychological deficits and learning problems were identified following perinatal brain injury. For example, severity of periventricular brain injury in preterm neonates is predictive of performance on a range of cognitive, motor, and behavioral measures in preschool and school age children.45-47

To address the question of relative impact of neonatal brain injury, we conducted comprehensive neuropsychological and neurobehavioral assessment of 152 neonates treated with ECMO and compared outcome at age 5 years according to severity of brain injury on neonatal neuroimaging. The outcome measures included intellectual status, preacademic skills, neuropsychological deficits, and neuromotor dysfunction. Details of the study have been reported elsewhere.41

The ECMO study patients met institutional criteria for ECMO and underwent VA bypass (the vessels were not reconstituted), had routine cranial ultrasounds daily during bypass and a CT scan within 3 weeks of decannulation. The neuroimaging findings were reviewed by a pediatric radiologist (DB) who was not aware of the neurodevelopmental outcome. Abnormalities were identified in 42% of the ECMO cohort and scored for extent and severity. Four groups were formed from the neonatal neuroimaging severity score: No lesion (N = 88), Mild lesion (N = 38), Moderate lesion (N = 12), and Severe lesion (N = 14) (See Table 1).

Fifty-three 5-year-old children who had not spent time in a special care nursery, were given the same neurodevelopmental test battery and served as a normative group for data analysis. They were comparable to the ECMO cohort in terms of birth weight, gender, ethnicity, mother’s marital status, and level of education achieved, home environment status, and age at test. The control children did not have imaging and were assumed to have normal brain structures.

The assessment protocol included a complete neuropsychological assessment, standard neurological evaluation, and assessment of gross motor and fine motor function. The neuropsychological battery included administration of six subtests of the Wechsler Preschool and Primary Scale of Intelligence-Revised (WPPSI-R short form) and additional subtests to assess function in six neuropsychological domains: receptive and expressive language, verbal memory, visual memory, visual-perceptual/spatial, visual-perceptual/motor, and attention/executive function. In addition to the standard neurological examination, four psychometric measures of left-side and right-side motor function included: balance (duration on either foot), grip strength (hand dynamometer), dexterity (pegboard), and fine motor speed (finger tapping). Mental disability was defined as mental retardation (full scale IQ < 70) or severe learning disability (full scale IQ < 70 but either verbal or performance IQ ≥ 80). Motor disability was defined by presence of paresis or reflex and tone abnormalities on the neurological examination, accompanied by motor function that was 2 or more years behind age level (Gesell Developmental Schedule).

One or more major disability conditions occurred in 26 of the 152 ECMO children (17%); 21 (14%) tested in the mentally retarded range or had severe learning disability; 8 children (5%) had a motor disability. One control child (2%) tested in the range of mild mental retardation. None of the normal control children had an abnormal neurological examination, but equal proportions of the ECMO and control

![Figure 4](https://example.com/image.png)  
**Figure 4** Severity of neonatal neuroimaging abnormality and rate of disability at age 5 years (N = 152). Control children (N = 53) had no neuroimaging, so normal brain structure is assumed.
children were found to have “suspect” neurological findings. As predicted, major disability at age 5 years was a function of neonatal neuroimaging abnormality (Fig. 4). We also computed the odds ratio and 95% confidence level for disability at age 5 years for each neuroimaging subgroup compared with the normal neuroimaging ECMO group (Table 2). The risk for handicap following mild brain injury was only slightly elevated compared with ECMO children who had normal CT scans, but both of these groups are at greater risk relative to the normal control children.

Severity of neonatal neuroimaging was significantly associated in a near-stepwise function with decreased intellectual status, greater neuropsychological deficits, and poorer preacademic skills (Fig. 5). Subtle effects of mild brain injury are again evident. Figure 5 shows lower IQ scores for ECMO children with mild brain injury relative to ECMO children with no identified brain injury. In turn, both of these ECMO subgroups are showing mild but consistent functional deficits relative to the normal control group (control group depicted as z scores = 0). Performance on psychometric measures of neuromotor function also showed a relative impact by injury severity (Fig. 6), but only the grip strength (left hand and right hand) and dexterity (left hand) reached statistical significance (P < 0.05).

Although prediction of neurodevelopmental deficit is of clinical significance, this body of research also shows evidence of compensation following even moderate to severe brain injury. Six of the 14 (43%) children in the severe neuroimaging group and 8 of 12 (67%) in the moderate brain injury group were not disabled. The full scale IQ scores of these 14 nondisabled children were all in the normal range or above (84-127). Evidence of normal, or near normal, functioning after significant structural brain damage in the perinatal period has been generally attributed to the relative neurolasticity of the immature brain, although symptoms may be latent.

Right Hemisphere Vulnerability of ECMO Neonate

Whether or not the neuroimaging data sufficiently support increased structural vulnerability to the right hemisphere in ECMO-treated neonates, some evidence of increased right-hemisphere vulnerability is present at a functional level. The preliminary question, however, is whether there is sufficient neurospecificity in the neonate in regard to neuropsychological or motor functions.

Hemisphere Specialization in the Neonate

Unilateral brain injury that occurs in older children and adults is followed by a predictable pattern of neuropsychological dysfunction; that is, language deficit after left hemisphere injury and visual pattern analysis deficit after right hemisphere injury. The contralateral hand may or may not be affected. When the unilateral brain injury occurs in the neonatal period, the pattern of deficit has not been as predictable when using global measures, such as verbal IQ and performance IQ. However, selective deficits in receptive and expressive language, visual/motor integration, and behavior problems have been identified in early childhood following unilateral brain injury during the perinatal period.48-50 Nota-

### Table 2 Odds Ratios for Disability at Age 5 Years by Severity of Neonatal Neuroimaging, Relative to Normal Control Children

<table>
<thead>
<tr>
<th>Severity of Abnormality</th>
<th>N</th>
<th>Abnormal (%)</th>
<th>OR</th>
<th>95% CI</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal Control</td>
<td>53</td>
<td>1 (1.89)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>88</td>
<td>9 (10.23)</td>
<td>5.924</td>
<td>0.73-48.16</td>
<td>0.090</td>
</tr>
<tr>
<td>Mild</td>
<td>38</td>
<td>5 (13.16)</td>
<td>7.879</td>
<td>0.88-70.46</td>
<td>0.078</td>
</tr>
<tr>
<td>Moderate</td>
<td>12</td>
<td>4 (33.33)</td>
<td>26.00</td>
<td>2.57-263.06</td>
<td>0.003</td>
</tr>
<tr>
<td>Severe</td>
<td>14</td>
<td>8 (57.14)</td>
<td>69.33</td>
<td>7.351-653.91</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

![Figure 5](image) Brain injury severity on neonatal US/CT scan and neurocognitive outcome at age 5 years (N = 152). Z-scores computed from normal control data (N = 53), thus control group mean = 0. FSIQ = Full Scale IQ. VIQ = Verbal IQ. PIQ = Performance IQ. FKSB = Florida Kindergarten Screening Battery (Adapted from Glass and coworkers, 1997).

![Figure 6](image) Brain injury severity on neonatal US/CT scan and neuromotor function at age 5 years. Z-scores computed from normal control data (N = 53), thus control group mean = 0. Dext. = dexterity (modified Purdue pegboard) Speed = rate of finger tapping.
bly, the effect of the side of lesion was consistent with the predicted pattern in adult neuropsychological studies.

To address this issue, we identified 24 children in our 5-year-old ECMO cohort who had neuroradiographically documented unilateral neonatal brain injury, and compared the performance at age 5 years of those who had unilateral neonatal left-sided lesions (N = 12) with those who had unilateral neonatal right-sided lesions (N = 12) on selected tasks reported to be predominantly mediated by the left hemisphere (language comprehension and production and right hand function) or by the right hemisphere (visual pattern discrimination and left hand function). Three specific tests from the neuropsychological test battery were selected. The Token Test is a measure of language comprehension (Lang/Comp) which requires the child to carry out with disks of different color, shape, or size a series of instructions that increase in syntactic complexity. Poor performance is generally accepted as a marker of left hemisphere functional deficit.48 Rapid Automatized Naming (RAN) is a timed test that requires the child to produce word labels (Lang/Prod) for a repeated set of pictures of common objects. The RAN is also sensitive to left-hemisphere deficit.31 Visual pattern discrimination32 requires the child match an abstract visual pattern to one of four choices which increase in complexity (Visual/ Discrim). As a nonverbal measure of visual/spatial function it is likely mediated by the right hemisphere.

We reported a significant association between the pattern of neuropsychological deficit (language versus visual/spatial) and the side of lesion, such that 75% of the Left-lesion group were more likely to have a lower Language/comp score than Visual/Discrim score; and conversely, 81% of the Right-lesion group were more likely to have lower Visual/Discrim score relative to their Lang/Comp score.50 The pattern was similar for language production, but not statistically significant. Excluding the one hemiparetic child, comparison of the individual difference scores between the right and the left hand for grip strength, rate of finger tapping, and dexterity (pegboard) revealed no reliable association between side of lesion and lateralized deficits. Therefore, on specific neuropsychological functions, but not psychomotor functions, the hypothesis of hemisphere specialization in the neonate was supported by the data.

**Evidence of Right Hemisphere Vulnerability at Age 5 Years Among ECMO-Treated Neonates**

There is some evidence for right hemisphere functional vulnerability among ECMO-treated neonates. We found an increased rate of right-hand dominance (94%) among our 5-year-old ECMO cohort, compared with our control group and to the normal population (85%). (This is also in contrast to an increased rate of left-hand dominance generally reported in high risk populations.) In addition, in comparing the left hand and right hand performance on psychometric measures, there is a suggestion of poorer performance by the ECMO-left hand, relative to the control group (z = 0) versus the ECMO-right hand, relative to control (z = 0). This is irrespective of the side of injury.

The larger question has been whether there is a particular neuropsychological profile of right-hemisphere vulnerability for the ECMO-treated neonate. The data demonstrate that the neuropsychological profile of the ECMO cohort seems to vary according to brain injury severity, rather than a dominance of functional deficits associated with the right hemisphere (Fig. 5). For example, ECMO children with no evidence of neonatal brain injury had a neuropsychological profile parallel to the normal control children. ECMO children with mild brain injury showed relative vulnerability on verbally mediated tasks. ECMO children with moderate-to-severe injury had a greater deficit in visual-perceptual and visual-motor tasks, relative to verbally mediated tasks. This profile of language vulnerability following mild brain injury and greater deficits in visual/spatial and visual/motor skills for children following moderate/severe brain injury is also reported in other brain-injured patient populations. Therefore, general findings of visual/perceptual or performance IQ deficits among children treated with ECMO are apt to be associated with moderate-severe brain injury, or perhaps a focal right-sided lesion, rather than a general right-hemisphere functional vulnerability.

Finally, as previously mentioned, there are frequent reports of widened interhemispheric fissure (IHF) found on CT after ECMO decannulation, although the clinical significance has not been systematically studied.20 Lago and coworkers reported significantly lower Bayley mental index and motor index at 6 and 12 months for a small group of ECMO-treated neonates who had enlarged cerebrospinal fluid spaces, but the location of the enlargement was not specified and may have included more generalized enlargement.18 Eight of our ECMO cohort at age 5 years had post decannulation CT scan evidence of widened interhemispheric fissure (in 7 of those patients it was the only finding). The mean Full Scale IQ score was 84 for these children, and all 8 of them had lower Verbal than Performance IQ scores. The sample is too small to draw firm conclusions, but the presence of widened IHF may not be benign.

**Summary**

ECMO-treated neonates incur a relatively high frequency of abnormalities identified on routine neuroimaging, which vary widely in type and severity. Multiple risk factors are present before and during cannulation which primarily affect cerebral blood flow. Brain injury severity identified in the neonatal period is predictive of neuropsychological status at age 5 years. Even those children with apparently mild degrees of injury appear to have some increased risk for neuropsychological deficits relative to normal control children. Right-hemisphere vulnerability is arguable at the structural level and only subtle in motor function. Greater vulnerability of right-hemisphere brain function is also reported in non-ECMO populations having
moderate-severe brain injury. Finally, compounding the effect of brain injury severity are other factors associated with poorer neurodevelopmental status, including pre-ECMO diagnosis other than meconium, lower birth weight, presence of chronic lung disease after ECMO therapy, and socioeconomic status.

References

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